Multiple monomorphic ventricular tachycardias in a structurally normal heart: A case report

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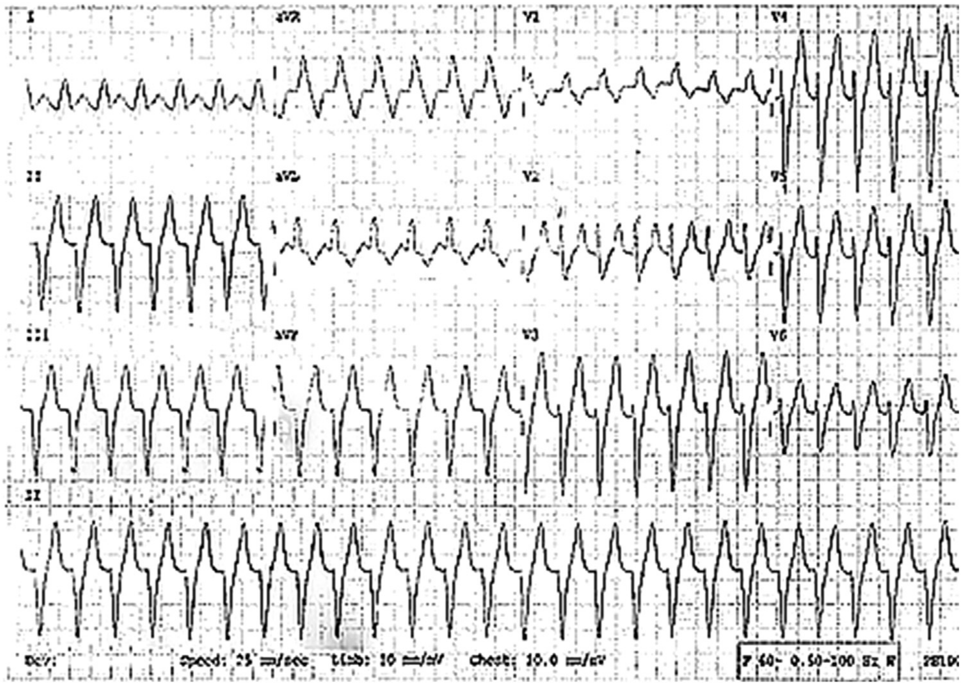

Figure 1 Surface ECG of clinical ventricular tachycardia with right bundle branch block and left-axis morphology.

Introduction

Catheter ablation has curative potential in patients with idiopathic ventricular tachycardia (VT). Different endocavitary structures, such as papillary muscles, moderator band, and false tendon, have been described as the substrate for idiopathic VT.[1–4] Although single-morphology VT arising from a false tendon is a well-known entity, multiple monomorphic VTs requiring ablation of a false tendon have not been reported. We report the case of a patient with multiple monomorphic VTs induced in the electrophysiology (EP) laboratory, which were successfully ablated from the left ventricular false tendon under intracardiac echocardiographic (ICE) guidance.

KEYWORDS False tendon; Fascicular ventricular tachycardia; Intracardiac echocardiography; Idiopathic ventricular tachycardia

ABBREVIATIONS **EP** = electrophysiology; **ICE** = intracardiac echocardiography; **LBBB** = left bundle branch block; **RBBB** = right bundle branch block; **RV** = right ventricle; **VT** = ventricular tachycardia

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Case report

The patient was a 28-year-old man with a 16-year history of symptoms of episodic palpitations despite calcium channel blocker and beta-blocker therapy. Transthoracic echocardiography was normal. Multiple episodes of wide QRS tachycardia with right bundle branch block (RBBB) and left-axis morphology were documented (Figure 1). He was referred to our institute for EP study and catheter ablation.

Mapping catheters were inserted via the right and left femoral veins. Multipolar catheters were positioned in the high right atrium, His-bundle region, and right ventricle

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KEY TEACHING POINTS

- All clinical ventricular tachycardias (VTs) in a structurally normal heart with right bundle branch block and left-axis morphology could not be attributed to classic fascicular VT. Different endocavitary structures such as papillary muscle and false tendon could be the site of origin.
- Although a false tendon is a single structure, it can give rise to multiple-morphology VTs.
- Intracardiac echocardiography can provide an additional advantage by identifying the actual site and extent of structures such as a false tendon and contribute to successful catheter ablation by optimizing tissue–catheter contact.

(RV). Tachycardia was not inducible by atrial and RV burst and programmed extrastimulation without and with isoproterenol infusion. When programmed extrastimulation was repeated from the left ventricle on isoproterenol, monomorphic VT with 5 different morphologies including clinical VT (of RBBB with left axis) could be induced: RBBB with northwest axis, RBBB with right axis, left bundle branch block (LBBB) with right axis, and LBBB with left-axis morphology. Of interest, all VTs had the same cycle length (270 ms). A 7Fr, 3.5-mm tip, open-irrigated tip catheter (Biosense Webster, Johnson & Johnson, Diamond Bar, CA) was used for mapping and ablation. The study was carried out under CARTO 3D electroanatomic mapping guidance (Biosense Webster). During activation mapping, VT terminated spontaneously and changed to a different morphology but with the same cycle length. ICE imaging was performed using an 8Fr linear phased-array imaging probe (Siemens, Berkeley, CA) placed in the right atrium and RV via the right subclavian vein. The left ventricular false tendon was visualized traversing the left ventricular cavity from the lower septum to the free wall (Figure 2). Radiofrequency ablation was performed at both the septal and free-wall insertion sites of the false tendon under ICE imaging (Figure 2). Figure 3 shows recording during one of the tachycardias, which terminated during ablation. The rest of the ablation was performed during sinus rhythm guided by pace mapping and ICE imaging. Postablation, the left ventricular false tendon was found to be intact by ICE imaging, and no VT could be reinduced using the same stimulation protocol. The patient has now completed 13 months of follow-up, off antiarrhythmic drugs and without any recurrence.

Discussion

It was previously believed that idiopathic VTs, especially RBBB with left-axis morphology, originate only from the fascicles. The advent of technologic breakthroughs, such as EP study and 3-dimensional mapping, along with the varying response of VTs to ablation have improved our understanding of the foci of idiopathic VTs. Apart from the fascicles, other endocavitary structures such as papillary muscles, false tendon, and moderator band have now been

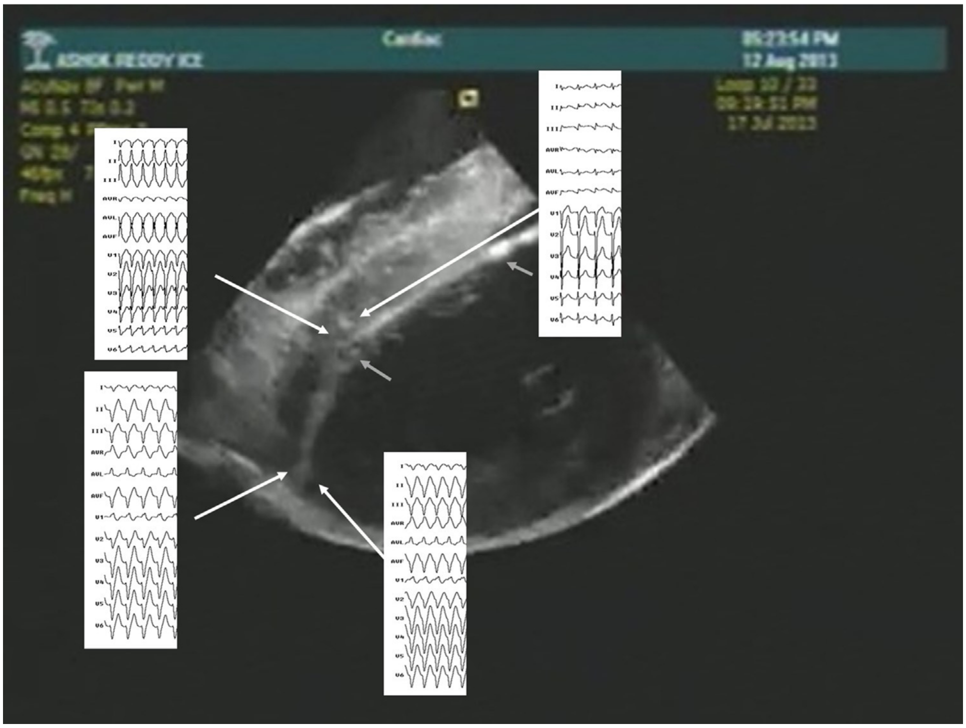

Figure 2 Intracardiac echocardiographic long-axis image showing false tendon extending from septum toward posterior wall of left ventricle. Ablation catheter *(gray arrows)* is seen at the junction of the septum and false tendon. The false tendon is a single substrate with multiple exit sites, explaining the different morphologies of ventricular tachycardia having the same cycle length.

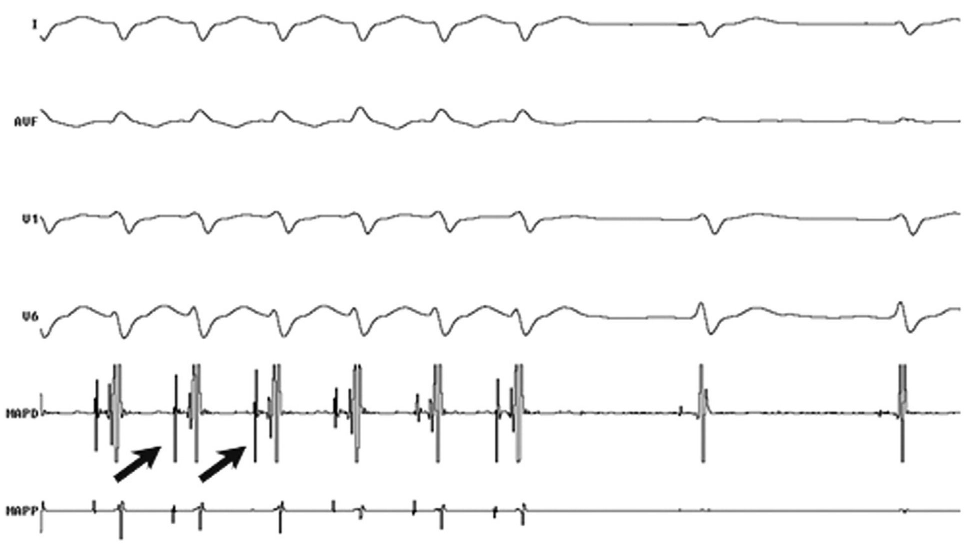

Figure 3 Surface ECG leads I, aVF, V_1, and V_6 and intracardiac signals. *Arrows* indicate presystolic potentials during successful site of ablation of LBBB with right axis morphology VT. Ablation catheter is located at the site of septal attachment of the false tendon. LBBB = left bundle branch block; MAPD = mapping catheter distal; MAPP = mapping catheter proximal; VT = ventricular tachycardia.

recognized as the cause of idiopathic VTs.[5,6] VTs arising from these structures usually produce single-morphology VTs, but in rare instances different morphologies have been observed.[3] The case described here is a rare observation in which the false tendon was the focus of VT.

VT arising from a false tendon generally produces a wide complex VT or fascicular VT with typical bundle branch block morphology. There have been reports of false tendon–related VT with polymorphic[7] or narrow QRS tachycardia morphology.[8] Our case exhibited multiple monomorphic VTs. The clinical VT in this patient had RBBB morphology with left-axis deviation, which is classic for fascicular VT. The same was induced as the first VT during EP study. This morphology usually is assumed to be reentry and can be entrained. However, entrainment was unsuccessful for all the VTs induced in the EP laboratory. Thus, the inability to entrain any of these VTs, the termination and spontaneous reinduction of VT with different morphologies while mapping, and the lack of fractionated potentials at the site of ablation all pointed toward a focal rather than a reentrant mechanism for the VTs. Different morphologies but the same cycle length in the VTs suggested a single origin but different exit sites. In this patient, radiofrequency ablation at both the septal and free-wall attachment sites was required to eliminate all VT morphologies. Apart from understanding the VT mechanism, stable catheter contact with the false tendon was an important contributor to the success of ablation. Thus, along with 3-dimensional mapping, ICE imaging was critical for optimization of tissue–catheter contact and successful ablation of multiple-morphology VTs.

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